08/896053

7/17/96

3/3,AB/2 (Item 2 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

14005715 BIOSIS Number: 01005715
Effects of in vivo adventitial expression of recombinant endothelial nitric oxide synthase gene in cerebral arteries
Chen A F Y; Jiang S-W; Crotty T B; Tsutsui M; Smith L A; O'Brien T; Katusic Z S
Dep. Anesthesiol., Mayo Clinic, 200 First Street SW, Rochester, MN 55905, USA
Proceedings of the National Academy of Sciences of the United States of America 94 (23). 1997. 12568-12573.
Full Journal Title: Proceedings of the National Academy of Sciences of the United States of America
ISSN: 0027-8424
Language: ENGLISH

13160715 BIOSIS Number: 99160715
Adenoviral-mediated transfer of the human endothelial nitric oxide synthase gene reduces acute hypoxic pulmonary vasoconstriction in rats
Janssens S P; Bloch K D; Nong Z; Gerard R D; Zoldhelyi P; Collen D
Cent. Transgene Technol. Gene Ther., KU-Leuven, Campus Gasthuisberg, 49 Herestraat, B-3000 Leuven, Belgium
Journal of Clinical Investigation 98 (2). 1996. 317-324.
Full Journal Title: Journal of Clinical Investigation
ISSN: 0021-9738

17/3,AB/14 (Item 14 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

12164999 BIOSIS Number: 98764999
Effect of recombinant endothelial nitric oxide synthase **gene expression on reactivity of isolated canine basilar artery**
Chen A F Y; Kinoshita H; Tsutsui M; O'Brien T; Pompili V J; Crotty T B; Katusic Z S
Mayo Clinic Foundation, Rochester, MN 55905, USA
FASEB Journal 10 (3). 1996. A303.
Full Journal Title: Experimental Biology 96, Part I, Washington, D.C., USA, April 14-17, 1996. FASEB Journal
ISSN: 0892-6638
Language: ENGLISH
Document Type: CONFERENCE PAPER

17/3,AB/18 (Item 18 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11983879 BIOSIS Number: 98583879
Expression and function of recombinant endothelial nitric oxide synthase **gene in coronary arteries**
Pompili V J; Stelter A; Smith L; Camrud A; Arndt L; Holmes D R Jr; Schwartz R S; Katusic Z S
Mayo Clinic, Rochester, MN, USA
Circulation 92 (8 SUPPL.). 1995. I295.
Full Journal Title: 68th Scientific Session of the American Heart Association, Anaheim, California, USA, November 13-16, 1995. Circulation
ISSN: 0009-7322
Language: ENGLISH
Document Type: CONFERENCE PAPER
Print Number: Biological Abstracts/RRM Vol. 048 Iss. 001 Ref. 013997

17/3,AB/16 (Item 16 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

12119008 BIOSIS Number: 98719008

Adenoviral **transduction of** endothelial **cell** nitric oxide synthase **gene yielding functional enzymatic activity in porcine coronary arteries**

Pompili V J; O'Bnrien T; Kullo I; Spector D; Schwartz R S; Holmes D R; Katusic Z S

Penn State Coll. Med., Hershey, PA, USA

Journal of the American College of Cardiology 27 (2 SUPPL. A). 1996. 290A.

Full Journal Title: 45th Annual Scientific Session of the American College of Cardiology, Orlando, Florida, USA, March 24-27, 1996. Journal of the American College of Cardiology

ISSN: 0735-1097

Language: ENGLISH

17/3,AB/7 (Item 7 from file: 5)

DIALOG(R)File 5:BIOSIS PREVIEWS(R)

13282148 BIOSIS Number: 99282148

Adenoviral-mediated transfer of human constitutive endothelial nitric oxide synthase **gene to rat lungs increases pulmonary nitric oxide production and inhibits platelet aggregation**

Nong Z; Hoylaerts M F; Zoldhelyi P; Gerard R; Collen D; Janssens S

Cent. Transgene Technology, Gene Therapy, Univ. Leuven, B3000 Leuven, Belgium

European Heart Journal 17 (ABSTR. SUPPL.). 1996. 98.

Full Journal Title: XVIIIth Congress of the European Society of Cardiology, Birmingham, England, UK, August 25-29, 1996. European Heart Journal

ISSN: 0195-668X

Language: ENGLISH

3/3,AB/2 (Item 2 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

14005715 BIOSIS Number: 01005715
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Chen A F Y; Jiang S-W; Crotty T B; Tsutsui M; Smith L A; O'Brien T; Katusic Z S
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Cent. Transgene Technol. Gene Ther., KU-Leuven, Campus Gasthuisberg, 49 Herestraat, B-3000 Leuven, Belgium
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Full Journal Title: Journal of Clinical Investigation
ISSN: 0021-9738

Author

19, 14, 12, 11, 1

(2) Setoguchi

(4) van der Leyen

7/3,AB/11 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

9816017 EMBASE No: 95372942
Endothelial **nitric oxide synthase in the lungs of patients with pulmonary hypertension (4)**
Xue C.; Johns R.A.; Giaid A.; Saleh D.
Virginia Univ. Health Sciences Ctr., Charlottesville, VA 22908 USA
New England Journal of Medicine (USA) , 1995, 333/24 (1642-1644)
CODEN: NEJMA ISSN: 0028-4793
LANGUAGES: English

R11.N4

7/3,AB/12 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

9801621 EMBASE No: 95335858
Persistent pulmonary hypertension of the newborn: Role of nitric oxide
Kourembanas S.
Joint Program in Neonatology, Children's Hospital, 300 Longwood Ave, Boston, MA 02115 USA
Journal of Intensive Care Medicine (USA) , 1995, 10/6 (270-282)
CODEN: JICME ISSN: 0885-0666
LANGUAGES: English SUMMARY LANGUAGES: English

NIS

Persistent pulmonary hypertension of the newborn (PPHN) is a common cause of respiratory failure in the full term neonate. Molecular and cellular studies in vascular biology have revealed that **endothelial** -derived mediators play a critical role in the pathogenesis and treatment of PPHN. **Endothelial** derived vasoconstrictors, like endothelin, may increase smooth muscle cell contractility and growth, leading to the physiologic and structural changes observed in the pulmonary arterioles of infants with this disease. On the other hand, decreased production of the **endothelial** -derived relaxing factor, nitric oxide, may exacerbate pulmonary vasoreactivity and lead to more severe pulmonary hypertension Exogenous (inhaled) nitric oxide therapy reduces pulmonary vascular resistance and improves oxygenation. The safety and efficacy of this therapy in reducing the need for extracorporeal membrane oxygenation and decreasing long-term morbidity is being tested in several trials nationally and abroad. Understanding the basic mechanisms that regulate the gene expression and production of these vasoactive mediators will lead to improved preventive and therapeutic strategies for PPHN.

7/3,AB/14 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE

9716668 EMBASE No: 95273228
Endothelium-derived vasoactive factors in hypertension: Nitric oxide and endothelin
Nava E.; Luscher T.F.
Cardiology, University Hospital, CH-3010 Bern Switzerland
Journal of Hypertension, Supplement (United Kingdom) , 1995, 13/2 (S39-S48)
CODEN: JHSUE ISSN: 0952-1178
LANGUAGES: English SUMMARY LANGUAGES: English

NIS

Endothelium-derived nitric oxide: The endothelium is a source of vasoactive factors among which the most relevant are nitric oxide and endothelin. Nitric oxide is synthesized from L-arginine by a family of nitric oxide synthases and is a widespread biological mediator. It is implicated in many physiological and pathophysiological processes, including a variety of cardiovascular diseases like hypertension. Nitric oxide and hypertension: The release of nitric oxide seems to be modulated by changes in blood pressure. However, the role of nitric oxide in hypertension is still controversial and seems to vary depending on the

stage of the disease and the model studied. In spontaneous hypertension, the production of nitric oxide is increased but inefficacious, probably because of increased inactivation or scavenging. In the heart the production of nitric oxide seems to be increased, probably as a compensatory mechanism against hypertension. In salt-induced hypertension, nitric oxide production may be impaired. In human hypertension, pharmacological experiments reveal an impaired nitric oxide dilator mechanism. In pulmonary hypertension, the use of nitric oxide gas inhalation has been proposed as a future therapy for this condition. Endothelin: Endothelin-1 is a potent vasoconstrictor peptide produced and released from **endothelial** cells. In isolated blood vessels, endothelin causes profound contraction. The hemodynamic effects of endothelin can be explained by the activation of two endothelin receptors, ETA and ETB. The relationship between endothelin and hypertension is not clear. Although plasma endothelin levels are normal in most patients with essential hypertension, the hypertensive blood vessel wall may contract more profoundly in response to the peptide; hence, endothelin antagonists may have antihypertensive effects in patients with hypertension.

7/3,AB/19 (Item 12 from file: 73)
DIALOG(R)File 73:EMBASE

NIS

9238280 EMBASE No: 94184787
Role of endothelial **-derived nitric oxide in normal and hypertensive pulmonary vasculature**
Archer S.; Hampl V.; McKenzie Z.; Nelson D.; Huang J.; Shultz P.J.; Weir E.K.
111C, VA Medical Center, 1 Veterans Drive, Minneapolis, MN 55417 USA
SEMIN. RESPIR. CRIT. CARE MED. (USA) , 1994, 15/3 (179-189)
CODEN: SRCCE ISSN: 1069-3424
LANGUAGES: English SUMMARY LANGUAGES: English
The role of EDNO in the PHT has been primarily evaluated in animals, with few human studies. Most research has focused on the chronic hypoxic form of PHT. With these limitations in mind, we conclude that basal pulmonary EDNO synthesis is preserved or increased in PHT. Although this EDNO up-regulation seems to reduce the severity of PHT, its relative importance among other modulating factors remains to be established.